A Clinical Lecture

ON

CHRONIC SPINAL MENINGITIS:

ITS DIFFERENTIAL DIAGNOSIS AND SURGICAL TREATMENT.

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THIS afternoon I desire to deal with a subject on which as yet I have published nothing, but on which I have been threatening to publish something for the last ten or twelve years. It is the question of operating in a group of cases which are apparently fairly common. For instance, without much trouble Dr. Grainger Stewart has collected (November, 1907) the records of 21 cases which I have operated upon. So it is a class of case which is met with fairly frequently, certainly more commonly than tumour of the spinal cord. It presents the same symptoms—except certain minutiae—as tumour of the spinal cord; indeed, these cases have usually been diagnosed as tumours of the spinal cord and transferred to the surgeon, and yet they are not tumours at all. They are cases of chronic spinal meningitis, the causation of which has yet to be finally determined. They yield to surgical treatment when ordinary medicinal treatment has failed. I do not mean to say that they all yield to surgical treatment—I wish they did—but so many do that it is quite clear that earlier diagnosis would have saved, in my opinion, the majority, if not all, of the cases.

Like all these subjects when a new clinical condition is recognized, many things are brought in under the same heading which do not belong to it at all; in fact, the very expression I have used—"chronic spinal meningitis"—would suggest to you at once that such a vague clinical expression must include a number of conditions which do not owe their existence to the same cause. The class of case I refer to you will recognize from my having said that they resemble tumour of the spinal cord. They are those in which an adult person—because I have only one case below the age of puberty—begins to complain of pain and progressive loss of power in the legs, with it may be also slight kyphotic curvature of the spine, and develops ultimately a progressive paraplegia that runs through the ordinary course and terminates fatally.

The first clear case of this kind which I met with was some ten years ago, when Sir William Gowers referred to me a gentleman who had complete paraplegia, who had a distinct but a very slight kyphotic curve of the dorsal vertebrae, and who, Sir William Gowers thought, was probably a case of caries and secondary paraplegia, which was commencing when he first saw the case but which was very advanced when the patient came under my care. I confess that, for my own part, I did not accept the diagnosis of caries, but I was unable at that time to substitute any other for it; and, inasmuch as the patient had compression paraplegia, I, of course, agreed to operate, from the general point of view of relieving the compression. But when I came to operate, I found that instead of the neural canal being narrowed by disease of the bone, it was narrowed because the theca of the cord was enormously distended and filled up the lumen of the canal. Further, on opening the theca, instead of finding a tumour inside, compressing the cord, I found nothing but a very considerable excess of cerebro-spinal fluid, and a cord that was rather shrunken. Being anxious lest there might be a tumour other than at the point exposed, I explored with a probe up and down the neural canal for a distance of 5 or 6 in. but found nothing. But inasmuch as the localization diagnosis of the upper limit of compression was quite clear, the symptoms corresponding to a lesion at the sixth dorsal root level, it was obvious there was nothing more to be done than to close the wound, and this I did. The patient made an ordinary recovery and gradually began to improve, until now, from having retired from the city, he has gone back to the city and is engaged in Stock Exchange work.

Such was the first case of the kind I have seen, and it attracted my attention very forcibly, because in the first instance an accurate diagnosis was completely wanting; and, secondly, because here was a condition which was getting worse under antisyphilitic remedies and every other kind of drug treatment, but the progress of which was completely stopped by laminectomy, freely opening the theca and washing out with mercurial lotion. The patient, as I say, got well, went back to work, and has remained well.

Without wearying you with details, I may say that I have seen a relatively large number of such cases, on most of which I have performed laminectomy and subdural mercurial irrigation. All the cases also which have been referred to me have been diagnosed at the time I have received them as tumour of the spinal cord or disease of the vertebral column. But naturally during ten years one becomes more wary, and I have for a long time shown that there are certain features in these cases which should lead to their being recognized as cases of chronic spinal meningitis and not tumour. That is the position which now deserves discussion. I would have raised this question long ago but for the fact that inasmuch as the first twenty operations were not followed by death, no anatomical examination of the condition was forthcoming.

In 2 cases, however, we have obtained the spinal cord. In one the operation had been completed, and six weeks after the wound healed the patient died suddenly from syphilitic myositis—probably of the atrioventricular bundle. In the other no operation was done, and the patient succumbed to the paraplegia. In both a meningo-gliosis (sclerosis) was found. The spinal cord, so often seen at the operations to be shrunken, was markedly contracted in a tabes case, and exhibited microscopically typical glial sclerosis.

I shall not take up your time by going into a critical examination of the accounts of chronic spinal meningitis which have been given by the classical neurological writers, although the clinical investigation of these cases needs a great deal of further work to render the description in the textbooks definite and practically accurate. The real matters of interest relate to the circumstances under which the condition arises, which can be relieved by surgery, what is the proper surgical treatment, and what is likely to be the result.

Of recent years similar cases have been described in America and in Germany. The first actually published[1] was one under the care of Drs. Spiller and Musser and operated upon by Dr. Edward Martin, the patient making an excellent recovery. In April, 1907, Professor F. Krause made an interesting communication[2] to the German Surgical Society, and described 6 cases (out of 22 referred to him as tumour cases) in which the hydrops and meningitis serosa spinalis were demonstrable as the cause of the paraplegia. The condition was determined in 2 of the cases by *post-mortem* examination. Krause quotes Dr. H. Schlesinger,[3] who described the condition anatomically in 1898 in a case of paraplegia with necropsy. Professor Oppenheim,[4] who had examined Krause's cases clinically and treated them medically, also regarded the disease as an extensive meningitis.

Since this lecture was delivered (July, 1907), there has been published a further brief communication from Dr. Spiller,[5] in which he again draws attention to the case published in 1903, and, after saying that the condition cannot be distinguished clinically from tumour of the cord, advises earlier surgical intervention.

The cases of parasitic and other cysts referred to in his argument with regard to the mechanical conditions of pressure do not elucidate further the nature of the cases under discussion, and therefore, in the short time at my disposal, I shall confine myself strictly to what Spiller calls "circumscribed spinal meningitis."

It will now be evident how closely these cases resemble instances of tumour of the spinal cord. I might take any one of the series of notes which I have before me and outline it. Let me take the last which we had, and which was put down on the notes here as "Spinal tumour, extramedullary." That is the diagnosis from the medical side, and it is as recent as July of this year (1907). So you see that up to the present time our diagnosis requires a good deal of perfecting. This patient is now in the convalescent home, so I regret I cannot show her to you.

She was a single woman, aged 40, without anything particular in the family history. There was a previous personal history of anaemia, but nothing in the nature of a serious illness until 1901. For the last ten years she had very heavy work as a dressmaker, with much standing and kneeling. Six years ago she had a severe attack of influenza, when she was ill for a month with pains all over her; and very soon afterwards she began to complain of pains in the right loin, worse on movement, and a feeling as if the muscles of the thigh were tightening. In drawing your attention to the fact that this case is referable to influenza as the probable cause of the meningitis, I would remind you of the cases of meningitis described in 1890 and 1891 by Horatio Wood of Philadelphia, because several of these cases have appeared now to be residual cases of such spinal meningitis as was originally described by Wood. The pains spread gradually to the front and back of the right thigh, and during the last twelve months they have caused painful cramping and twitching of muscles of the right leg. The right toe often cocks up involuntarily with the pain. Ultimately, therefore, the pain in this case has affected the whole of the right leg, having begun in the right groin. It involves a diffuse area, one which is supplied by several nerves; there is no particular nerve-root distribution which is involved, but the whole limb is in pain, and can be shown on a chart. I invite your attention to this point first, because this I consider to be the most valuable among the minutiae which serve to distinguish these cases from cases of spinal tumour. A tumour of the spinal cord commonly exhibits the symptom of pain as localized to one nerve root; it does not give you, as a rule, areas of pain which necessarily involve many nerve roots. If spinal tumour cases are examined minutely it will be found that in the large majority of them the pain begins at or near one nerve root, and to the distribution of that spot the patient will chiefly ascribe his or her pain. This chronic spinal meningitis, on the other hand, being a general condition, tends to affect diffuse areas painfully, exception being always made for the upper limitation of anaesthesia. (See below.)

We will now pass on. For two years or more there has been besides this pain marked tightness and numbness of both thighs, as if they were made of marble. During the last two years there has been slight dribbling on micturition, and during the same period the pain has been so severe that it limited freedom of walking, so that the patient could only take one step at a time in going up and down stairs. Four weeks ago there was severe pain in the small of the back. Many of these patients describe a painful area in the spine without necessarily exacerbation on movement. On admission the patient was a very stout, healthy-looking woman, with no indication of disease in the circulatory or alimentary systems, nothing abnormal in the cerebral nerves, or cranium or upper limbs. The pupillary reactions were equal and normal. So to the middle of the body there was nothing discoverable. There was slight wasting of the whole of the right leg and general weakness of the whole of the right leg—see how universal these symptoms are—some eversion of the ankle with weakness on dorsiflexion of the foot, and some extension of the hip. There was very considerable alteration of sensation. The alterations of sensation were these: that she had relative anaesthesia of the whole of the right limb and of the right hypochondriac region, the anaesthesia being to all forms of sensation as well as relative analgesia. The coexistence of analgesia and anaesthesia I always think is an extremely serious point to bear in mind in all spinal cases, because it carries with it the assumption that the spinal cord itself is centrally involved in the lesion, whatever it be. With regard to the deep reflexes, the knee-jerk was markedly diminished on the right side, present on the left, but both were, on the whole, diminished. I will come back to the question of the knee-jerks later. As regards the superficial reflexes on admission, the plantar reflex on the left side is flexor, but it is less definitely so on the right. When I examined her later I found she had an extensor response. The abdominal reflexes were present on the right and left. With regard to the sphincters, I have said already that there had been dribbling after micturition. There should have been added there a most important point, namely, that there was no trophic lesion whatever in the skin, nor apparently any tendency to decubitus. On testing her with pilocarpin to map out the deficiencies of the secretory system, for comparison with the tactile anaesthesia, according to the methods which I have demonstrated as useful for twenty years, there was found a very marked parallelism.

That is the story of a typical case. On operating at the level of the dorsal segment a marked degree of chronic meningitis was found and relieved. But such extremely marked localization, one side being apparently chiefly affected, would of course lead to the assumption that certainly one half of the cord must be primarily affected, and might carry with it the further assumption that the lesion was more probably a tumour than general meningitis. I wish to draw particular attention to this point at this stage, because all these cases, if you look into them carefully, present a certain degree of unilaterality by the time they come under our direct notice. In further illustration of this point I show you sections of the spinal cord of an interesting case, which was not treated surgically and died in the hospital a short time ago. In these specimens you will notice that there was a condition of things exactly parallel to what I have frequently observed—namely, a sclerotic condition of the cord, beginning below and creeping up the cord. As a matter of fact, all the lower part of the cord shows only the endogenous fibres preserved. When you look at it with the lens you see that the ascending degeneration has affected one side notably more than the other. Consequently, although it is put down as universal disease, it nevertheless has a distinctly unilateral pathological locus. So the fact that we find that most of these cases have a unilateral origin must be recognized at the onset, because it must not prejudice our opinion. Probably we are naturally inclined, when there is marked unilaterality in the symptoms, to jump to the conclusion that it must be tumour. But now, after this series of cases, it must be recognized that that is not the fact—that it may equally be this condition of chronic general meningitis.

Now as regards further details. First the quality of the pain. As I said before, pain is felt in one limb to begin with, and then it spreads to the other limb, and then up the back. It is generally said by the patient that the pain is in the substance of the limb, and it feels in the majority of cases like a tightening or drawing up, and most patients complain specially of the sensation of tightness. And while speaking on that point I may also draw your attention to the fact that sometimes they have in addition the typical girdle tightness or compression, a feeling as of a band around the middle of the body. With regard to the degree of the pain, it may be similar to that in tumour cases, in that it may be so severe as to prevent sleep.

With regard to ordinary sensation, we will refer to this under the heading of "hyperaesthesia and anaesthesia." With regard to hyperaesthesia, this patient whose case I have just recounted to you on some days had very marked hyperaesthesia over the whole leg, a condition of things which I have never seen in a tumour case, in which at the outset there would be either a marked zone of hyperaesthesia at the upper limit of the anaesthesia, or a similar area corresponding to the nerve root or roots on which the tumour was situated. But when there is hyperaesthesia over a large area, it ought always to suggest to you that a number of nerve roots or their central representation mechanisms are in a state of irritation, at any rate in a state of physiological hyperexcitability. So that the distribution of the hyperaesthesia in these cases is an important symptom as helping to correct the diagnosis.

With regard to tactile anaesthesia, none of these cases that I have seen yet have had absolute anaesthesia; it has always been relative, and sometimes it has been present only to a slight degree, though extending over a large area. Microscopically, in neither of the two cases in which we have been able to obtain a *post-mortem* examination so far were the nerve roots much affected. So it is easy to understand that there may be widespread paraplegia, and yet no absolute anaesthesia anywhere. Nor is there dissociation, for as this is a general affection of the roots altogether, all the fibres are affected, and therefore all forms of sensation—touch, temperature, etc.—are affected in greater or less degree.

Next, as regards efferent phenomena. First, with regard to movement. How do these patients become paralysed? They become paralysed first by a gradual sense of weakness coming on in the whole leg. You know that is a very common description given to you by patients who become paraplegic from tuberculous caries, because in that condition you get a kind of ischaemia of the spinal cord as a rule, and the whole machine, as it were, becomes weak, the conductivity of the whole cord becomes impaired, and therefore the patient says the whole leg is weak. So it is with these cases, where there is a general condition affecting the spinal cord and its roots, naturally the symptoms are grouped *en bloc*. As can be seen from these notes of the cases, a localized extra weakness of one joint, as for instance the ankle or the hip or the knee, may often be seen, but no restricted root paralysis—that is to say, paralysis of groups of muscles served by any one root. The general loss or diminution of the action of one joint involves the decadence of several segments of the spinal cord. Thus a gradual weakness which develops until at last the patients cannot stand. In a certain number of cases, even in a comparatively early stage, they complain of spasticity. So that some of these cases undoubtedly are diagnosed as instances of lateral sclerosis—that is, the old idea of lateral sclerosis; and when you come to examine the spinal cord in a very severe case you will find there is not only a lateral sclerosis, but a general sclerosis throughout the cord. The explanation of the sclerosis I will come to later.

With regard to the other efferent phenomena, I have already referred to and demonstrated that secretion is proportionately paralysed with the advance of the paraplegia. With regard to the vasomotor system, as a rule these patients show no vasomotor phenomena, quite unlike a bad case of compression paraplegia from caries or tumour, where vasomotor phenomena are more often observed. One limb may become oedematous, the joint may fill up with fluid—a so-called myelitic arthritis—and where at one stage the part may be crimson in colour, and as the paraplegia develops it may change into pallor, and, finally, in the last stage there may be obvious vasomotor contraction extremely marked with a shrunken dry skin. These meningitis patients do not present this picture at all; their skin looks very normal, and, as I said before, they show no tendency to trophic disturbance. Possibly at the very end they ultimately develop bedsores, like other paraplegics, but they have not the striking tendency to decubitus that an ordinary case of localized compression of the cord has. Before passing from the question of sensory troubles, especially in connexion with the nerve roots, I must refer to one private case which illustrates very strikingly the connexion between affection of the roots and this general affection of the membranes of the cord. The case was that of a lady, whom I saw with Dr. Ferrier originally, and who had complained of a severe pain in the heel—that is to say, in that branch of the posterior tibial nerve which supplies the skin of the heel. This pain was for many years treated as a functional pain, and the unfortunate family differences which followed only emphasized once more the extreme necessity of caution in diagnosing functional disease. One day she suddenly developed a peculiar outbreak of herpes in the distribution of the first and second lumbar roots—that is, a definite root distribution; and then it was naturally thought that the posterior tibial trouble was a peripheral neuritis. When I saw her with this herpetic eruption it was quite obvious that she had what appeared to be an affection of the whole nerve supply of the limb, but I did not recognize at the time that she was one of this class of case. However, the pain was so great and disabling that I suggested dividing the posterior roots of the first and second sacral nerves. This was not accepted by those in direction of the case, and for two years this patient went on, gradually walking less and less, until at last she was practically sitting in a chair all day long. Still, she had no paraplegic symptoms of the ordinary kind. It was then decided that she should be operated upon, and accordingly I exposed the theca of the spine over the lower end of the lumbar enlargement, in order to divide these two roots. As I then found the theca enormously distended, of course it was apparent that the case must be one of chronic spinal meningitis. On opening the theca a great rush of cerebro-spinal fluid occurred, the lumbar enlargement and the commencement of the cauda equina were covered with thick yellowish-white matted arachnoid, and yet, as above stated, the pain was limited to the first and second sacral area, and the herpes to the first and second lumbar. Thus, the intermediate roots apparently had not suffered sufficiently to cause symptoms. I divided the posterior roots of the first and second sacral nerves, and then followed out the treatment I advise in these cases, and irrigated out the subdural space thoroughly with 1 in 1,000 sublimate solution. She made a very good recovery, and now she walks five miles at a time, and has, of course, lost her pain. It is now three and a half years since the operation was carried out, and she is apparently in perfect health, a position which is, perhaps, partly due to spinal mercurial inunction, which I have had continuously carried out since the operation. But this is the only case of the whole series I have seen in which there was a herpes, in which there was any evidence of posterior ganglionic affection, or any evidence of such minute root localization, and I only dwell upon it because it is such a striking illustration how in these cases we can be deceived.

Now we come to the question of the part of the spinal cord affected in this condition. This certainly is a very interesting question. The two cases I have used as illustrations obviously began in the region of the cauda equina, and in the large majority of these 21 cases the mischief apparently began all over the lower half of the back of the cord, and the large majority of them, I find on going over the notes, have (in consequence of the need of exposing the highest segment of cord symptomatically involved) been operated on at a point somewhere between the fourth and ninth dorsal—that is to say, in the mid-dorsal region. This is interesting, because, of course, we know that for one reason or another the mid-dorsal region has been, certainly for seventy years, recognized to be the usual seat of so-called transverse myelitis—at any rate, it has been referred to that region without any explanation being forthcoming. Whether it commences—like this case, for instance, which begins about the lower part of the lumbar enlargement and gradually goes up the cord—or whether it attacks the lower half of the cord simultaneously, will probably not be found out for a long time. But, however that may be, by the time the case is referred for operation, the exploration must begin in or just below the mid-dorsal region, because in the majority of these cases the zone of anaesthesia extends from the sixth to the eighth dorsal nerve root.

With regard to the age of the patients, I have already said that, with one exception, they were all adults. This question of age has a very important bearing, first on the question of the causation of the mischief, and, secondly, on the prognosis. I will therefore deal now with these two points. We will speak of prognosis first, because that is relatively an easier point. As we have to deal with a chronic inflammatory condition, the prognosis simply depends on the usual rule, that if a patient is below middle life or at middle life recuperation is likely to be very good; if beyond middle life, it is likely to be very poor. The oldest case which I find I have operated upon was a private patient, a lady who was 60 years of age, and I am sorry to say she did not improve in the least. It was a very bad case; there was a great deal of pachymeningitis, and there was a depressing history of congenital disease. She only had one brother alive and "healthy." One brother had died of locomotor ataxy, and a sister, whom I saw, suffered severely from combined syphilitic sclerosis, and so on. I believe the fact that she did not improve was entirely due to her age. Turning now to the next point—namely, the cause of the condition—I have said that there is a considerable excess of cerebro-spinal fluid in these cases, and have referred to the thickening of the arachnoid and the matting of the nerve roots. Before we discuss further the pathological conditions, I ought perhaps to make reference to the collection of cerebro-spinal fluid in the neural canal as a clinical fact, and discuss with you whether it has any bearing on these cases. The cerebro-spinal fluid, of course, is secreted by the lining membrane of the cerebral ventricles, by the lining surface of the choroid plexus, and it is secreted at a rapid rate. Although it is secreted under pressure, it is thus manufactured quickly, and if the pressure is relieved by a little trephine opening connected with the

ventricles or other part of the cerebro-spinal system, it escapes in very large quantity. There are several other points connected with the physiology of the cerebro-spinal fluid which are of interest. The ependymal epithelium, which is flat in the ventricles, becomes columnar at the commencement of the aqueduct of Sylvius under the posterior commissure, and this fact, as Professor Dendy has shown, applies to all animals, and is especially marked in the mouse and animals as low in the scale as that. So it would seem to have some physiological significance, and he suggests that the cilia of these epithelial cells serve by accelerating the circulation of the cerebro-spinal fluid downwards. Of course the cerebro-spinal fluid as it is being secreted must be got rid of; it must flow out of the ventricles, and must pass into the cavities of the body. It has been shown that as it is manufactured high up, under positive pressure, it finds its way out along the lymphatics along the course of the nerve roots. That it has this circulation in the cord is proved first by the injection of coloured fluids into the ventricle, and also by such very interesting pathological cases as the one published last year by Dr. Barnes, where a growth in the ventricle was disseminated all the way down the central canal, showing that the virus was gradually carried down along this natural highway, as it were, by the stream of cerebro-spinal fluid.

As regards the spinal cord, we find that in these cases there is a large collection of cerebro-spinal fluid around the cord, and it is under great pressure, because in the first place it distends the dura mater, so that it almost fills the canal, and there is no impulse in the theca when it is exposed for laminectomy. Instead of the theca rising and falling with respiration and also indicating the pulsation of the heart, there is no movement in it whatever. When you open the ballooned theca the fluid jets out, and then, when the whole thing collapses, you can see that there is a normal active movement of the cord; so that, at any rate, by the time these cases come to operation it is obvious that this physiological relationship of the cerebro-spinal fluid has become very abnormal, and that instead of having a proper circulation you have stagnation. I do not want to push this point too far, because we know so very little about it. We know nothing from the experimental point of view beyond the effect on the spinal cord of persistent stagnation or accumulation of cerebro-spinal fluid, but it is suggestive. And, seeing that these patients who do improve may improve so markedly simply from the free opening of the theca *plus* washing out—the effect of which I will discuss presently—it is very difficult to get away from the idea that this pressure does play an important part, and that consequently some of the benefit of surgical treatment is to be attributed to getting rid of such pressure.

The spinal cord, being subjected to this pressure, necessarily must undergo some ischaemic changes; and here we are in a very great difficulty, because we have no pathological material to enable us to say whether the changes which we can find in the only two cases which have died during these years are to be looked upon as truly representative because they were both cases in an advanced degree of the disease.

Causation.

That brings me to the next consideration—namely, the causation of the meningitis. In one of the cases just referred to certainly, and in both to some extent, there would be no doubt that syphilis was the cause; and, under those circumstances, one would say naturally that the sclerosis of the cord which was so very clear (see below), was a primary syphilitic sclerosis.

The point of chief interest is the condition of the cord in an average case. In every case when the theca has been opened and fluid allowed to escape the cord has presented a shrunken appearance, and once a slightly yellowish tinge. I have never operated upon an early case, except the one I referred to in which there was herpes, and there the matting of the arachnoid precluded an opinion as to the condition of the cord. I show you here the cord of one of the two cases referred to above, a man who was in this hospital, and on whom I operated some time ago. He had recovered from the operation and was commencing the usual improvement, when he died quite suddenly. The immediate cause of death proved to be the common event in cases of chronic syphilis—namely, syphilitic disease of the atrioventricular bundle of the heart. In this case the cord presents a remarkably flattened appearance, and on examining the sections with the lens there is seen a definite sclero-gliosis all round the periphery of the cord. In this case, I think, all the columns are universally affected except in the cervical region. The nerve roots themselves are very slightly attacked, and therefore the incoming fibres of the column of Burdach are not much affected, but the fibres of the column of Goll are particularly. Therefore this case, at any rate, was not simply an instance of pachymeningitis, but pachymeningitis combined with a certain degree of sclero-gliosis of the cord. It is quite possible that this was the pathological condition in the majority of the cases I have seen and operated upon, but it is impossible with the present means at our disposal to speak positively.

Since the difficulty just experienced of discussing accurately the pathology of these cases is due to the fortunate rarity with which laminectomy ends fatally, I may fitly conclude by describing their treatment. In every instance all these cases have been liberally treated with drugs, and have had the most conscientious anti-syphilitic treatment when referred for surgical care, and yet they have steadily gone downhill. This, by the way, might be advanced as an argument that they cannot have had their origin in syphilis. Although, on my own part, I think a number of them are not actually syphilitic, some of the cases certainly are, and, as so constantly happens in syphilis of the nervous system, resist all treatment not directly applied by surgical intervention.

On the question of syphilis, I said earlier in the lecture that though this chronic meningitis was a disease essentially of adult age, there was one case I had treated in which the patient was not an adult, namely, the instance of a boy aged 12, who was admitted into the National Hospital under Dr. Beevor three years ago. His history was that he had a rather rapidly oncoming difficulty in walking, with cramps in the right leg, followed by weakness, and then in the left leg. Later the legs jumped, but there was no girdle sensation and no sphincter trouble. When he was admitted he also had a slight degree of nystagmus, which was a cerebral point. He had spastic paresis in the arms and legs, slight loss of power, and so on. It therefore seemed to be a definite case of high affection of the spinal cord, and the nystagmus was the only point which might have led one to hold one's hand. But, of course, nystagmus in a child, especially with a slight visual defect, was not enough to establish a contrary diagnosis or one of cerebral complication, and when I came to expose the theca I found it was a typical case of chronic meningitis, the theca being notably distended and a large amount of cerebro-spinal fluid liberated. The cord appeared normal, and on examination of the posterior median fissure nothing abnormal was found. He went out greatly improved. I believe that was a case of congenital syphilitic origin (cf. Gilles de la Tourette). But the record of causation is like much clinical work, unfortunately, in being the expression of beliefs rather than actual scientific fact. For my own part, since so very few of these patients have suggested traumatism as a cause, it is not a likely one, and, as Oppenheim particularly states, such serious meningitis is not a sequela of concussion or railway spine. Then when we come to think of other kinds of chronic specific inflammation we shall conclude, I believe, that a certain number of these cases are gonorrhoeal. I am quite ready to admit that we have not proved cytologically or bacteriologically that the cerebro-spinal fluid contains either excess of lymphocytes or gonococci, but as regards excess of corpuscles, that is not a necessary concomitant when we think of gonorrhoeal arthritis. Therefore the cytology of the affection is of less importance, and, as regards the bacteriology, great difficulties exist in tracing the gonococcus. I regret that I have no direct evidence to substantiate my view, and the only point I can adduce is that infection with gonorrhoea occurred in several cases relatively a short time before the occurrence of the spinal symptoms and in the absence of other infections.

Treatment.

Now for the detail of this surgical treatment. I have already suggested to you in the remarks made earlier that

it consists in simple laminectomy, opening the theca, and washing out the theca with a mercurial lotion. Here we enter at once on the general and interesting question of notable benefit attending the free opening of the so-called serous spaces and washing out with some antiseptic, because I suggest the parallel of this surgical treatment of spinal meningitis is the well-known surgical procedure of opening the abdomen to cure tuberculous peritonitis. I believe there is no risk in washing out the theca with strong solutions of mercury, even up to 1 in 500 strength, though I have only used that strength once or twice. Still, I should not have any hesitation in using it at any time where there was much exudation in the arachnoid, following it up with 1 in 2,000, and leaving in some of this strength when closing the wound.* The skin wound, I think, should be completely closed, not drained. I used to sew up the theca, my object being to arrest the escape of cerebro-spinal fluid into the wound. I have now given that practice up in almost all cases, because in the first place I found wounds healed as quickly, and there is as a rule no greater escape of cerebro-spinal fluid, so that that reason for suturing was not a very important one. Undoubtedly, too, there is less discomfort to the patient afterwards—namely, less tendency to headache and less tendency to the temporary pyrexia and tachycardia which laminectomy cases not infrequently show. But I have also come to believe that allowing the escape of the cerebro-spinal fluid into the wound cavity for a time, for a few days at any rate, secures a valuable new state of things, for it must get away. It does not necessarily get away through your suture opening, which remains as dry as when closed; therefore it must drain away through the lymphatics of the walls of the wound. If we can thus establish an artificial exit for the cerebro-spinal fluid by means of the lymphatics in and among the muscles surrounding the spine, then, of course, we have established a fistulous association for the carrying on of the circulation of the cerebro-spinal fluid exactly analogous to Senn's and Cushing's methods of treating hydrocephalus. Non-suturing of the theca in the operation, therefore, has a possible advantage. It is difficult to prove that this circulation exists to the degree one believes, but at any rate most of the fluid escapes in that way necessarily because it does not flow out between the stitches of the closely sutured wound. After the wound is completely healed, free mercurial inunction of the spine should be ordered, especially over the scar, on the idea that having arrested the disease an attempt should be made to secure active "absorption" and a complete restoration to health.

In concluding this sketch of an imperfectly understood chronic condition I would express the view that probably many cases of so-called acute myelitis are really meningeal in origin, and that a laminectomy and free drainage of the subdural space might arrest the whole process and the subsequent fatal injury which the cord sustains in such conditions.

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*As a matter of fact the circulation of cerebro-spinal fluid is so active that probably any drug introduced into the theca is very soon washed out into the wound and lymphatics.

THE sanitary staff in Cuba has recently been increased with the view of mastering the yellow fever, which has not been under complete control, and the addition has proved a fortunate step, for last October was a particularly wet month. The rain in Havana was continuous, rendering the mosquito campaign most difficult. It is, moreover, in October that *Stegomyia* is most abundant. The magnitude of the task may be judged from the fact that the number of houses visited and "petrolized" during the month was 51,381, and that there were 176 cases in which *Stegomyia* larvae were found. There were 15 cases of yellow fever in July, but these dropped to 2 in August and 1 in September, while in October no cases occurred. The work of the staff is, however, by no means confined to the war against mosquito larvae. The dairies are kept under very strict inspection, and during the month of October alone 2,416 analyses of milk were made, and only 38 samples were found to be unsatisfactory.

THE SCIENCE COMMITTEE

OF THE

British Medical Association.

REPORT CXII.

OBSERVATIONS ON THE PHYSIOLOGY OF THE FEMALE GENITAL ORGANS.*

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I.—GENERAL CALCIUM METABOLISM.†

SINCE much of our work upon the physiology of the female genital organs has centred around the connexion existing between the calcium metabolism and the functions of these organs, it has been necessary for us to leave the bypath of our special investigation for the main road of general physiology, in order that we might, if possible, learn more than is usually known or understood concerning the rapid tissue changes which undoubtedly occur in the general calcium metabolism. We felt that unless we had some guide as to the ordinary (non-genital) influences at work we should be unable rightly to interpret or to assign proper importance to any observations we might make in connexion with the special functions under investigation.

We feel, at the same time, that it is expected of clinicians, when they leave their sphere of greatest activity, that they shall duly acknowledge and recognize the invidiousness of their position. We do this most assuredly, and most earnestly crave indulgence for the incompleteness and sketchiness of our work. In extenuation, it will be easily understood in the first place that we have been unable to devote continuous time to the work, which, consequently, has been carried on under difficulties; and, in the second place, that we have only endeavoured to get some insight into the general calcium metabolism in order to control our special observations. The real work on these general questions has yet to be done by others. We trust that some enterprising physiologists, to whom the work really belongs, will take it up—if, indeed, many of the matters to which we shall allude are not already under consideration.

In any investigation of the rôle of any product of, or factor in, metabolism, it is necessary first of all to be able to isolate that body from the tissues to which its activities are chiefly confined, and to be able to estimate any changes it may undergo quantitatively and qualitatively in different circumstances.

In order to be able to investigate the variations in the blood-calcium content, one of us (W. B. B.) devised a method for the quantitative estimation of the calcium salts. A preliminary communication was made in the BRITISH MEDICAL JOURNAL, April 20th, 1907, concerning this method and the reasons for which it was devised. Briefly, it consists in precipitating the calcium in the blood in the form of crystals of calcium oxalate, and, after certain dilutions and precautions against the formation of other crystals, counting those of calcium oxalate, which are very uniform in size, on a haemocytometer plate. Any slight inequality in size is corrected by the large

*The authors have been greatly indebted to the Science Committee of the British Medical Association for money grants. They also wish to express their indebtedness for assistance to Mr. George Barker, Mr. T. Williamson, and Mr. James Smith; also to Professors Benjamin Moore, Salvin-Moore, and Sherrington for laboratory facilities.

†This article is based on a paper read by W. Blair Bell, upon Some Observations on the Physiological Importance of the Calcium Salts, before the Liverpool Medical Institution, February 13th, 1908, and published in the *Liverpool Medico-Chirurgical Journal*, July, 1908.